2:30

798-3 Assessment of Stunned Myocardium by Dobutamine Echocardiography: Relation Between Myocardial Contractility and Distal Coronary Flow

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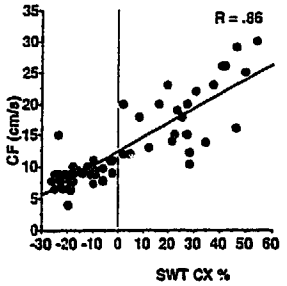
The contractile response of stunned myocardium to Dobutamine (D) infusion is variable and includes, a) biphasic response (improvement at low dose followed by worsening at high dose) or b) continuous improvement in contractility throughout D infusion.

In order to assess the relationship between the myocardial contractile response and distal coronary flow (CF), 10 mongrel dogs were instrumented. A circumflex (CX) stenosis severe enough to induce wall motion abnormalities at rest was created. D was progressively infused up to a rate of 40 mcg/kg/min. Coronary flow was assessed with a Doppler-tipped flow wire placed distal to the stenosis. Two dimensional regional systolic wall thickening (SWT) and distal CF was obtained at each stage of D infusion in the CX supplied territory.



A biphasic response was observed in 5 dogs. Continuous improvement in SWT was observed in 3 dogs. Two dogs had no change in their SWT throughout D infusion.

A very strong and positive correlation was found between the regional SWT in the circumflex territory and coronary flow distal to the stenosis.

The contractile response of stunned myocardium to progressive Dobutamine infusion is variable and parallels changes in distal coronary flow. The amount of available coronary flow distal to the stenosis, determines the contractile response of the myocardium.

2:45

798-4 Use of Myocardial Contrast Echocardiography to Study the Relationship Between Response to Dobutamine Stimulation of Asynergic Segments and Regional Perfusion Abnormality

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Combined use of dobutamine stress echocardiography (DSE) and myocardial contrast echocardiography (MCE) offers a unique possibility to study the relationship between myocardial function and perfusion. To evaluate the relationship between dobutamine wall motion response and myocardial perfusion, we performed MCE (sonicated loxaglate) and DSE (3 up to 12 μg/kg/min.) during catheterization in 20 patients (110 segments) with ischemic left ventricular dysfunction. Wall motion was assessed in a 16-segment model and divided into four responses; biphasic (improvement at low and worsening at high dosage), improved, worsened and no change. Before dobutamine infusion, the presence (+) or absence (–) of myocardial perfusion was assessed from MCE in asynergic segments at baseline. Of 72 asynergic segments with enough myocardial perfusion, 40 segments showed biphasic response and 26 segments improved. Of 38 segments without myocardial perfusion, 8 segments worsened and 30 segments did not respond to dobutamine infusion. A lot of segments (92%) with myocardial perfusion initially improved and all segments with perfusion abnormality did not. Dobutamine responses in each type were as follows.

	Biphasic	Improved	Worsened	No change
Perfusion (+)	40	26	4	2
Perfusion (–)	0	0	8	30

In conclusion, a lot of dobutamine augmentation of asynergic segments well coincide with myocardial perfusion of MCE. The microvascular reserve from MCE may assess the extent of myocardial viability and predict the recovery of asynergic segments following revascularization.

3:00

798-5 Dobutamine-Induced ST Segment Elevation in Patients With Recent Myocardial Infarction: The Role of Myocardial Ischemia, Viability and Ventricular Dyssynergy

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Background: The meaning of a stress-induced ST segment elevation (ST-↑) in Ecg leads exploring recent Q-wave myocardial infarction is still controversial. To evaluate the role of wall motion abnormalities (WMA), myocardial ischemia (MI) and viability (MV) in pts with ST-↑ in the ECG during dobutamine echocardiographic test (DET), we analyzed a series of 391 DET performed 10 ± 2 days after a first AMI.

Methods: DET-induced ST-↑ was defined as 1 mm greater than baseline at 40 ms occurring in > 1 Q-wave lead. Resting wall motion score index (WMSI-0) was derived using a 16 segment-4 grade score model. Homozonal MI was defined as worsening or new WMA in infarcted related artery perfusion territory. MV was defined as improving resting WMA in at least two infarct related segments during low dose DET. The extent of MI and MV was derived calculating the increase and the reduction of WMSI-0 (↑-WMSI and ↓-WMSI) at peak stress and at low doses, respectively.

Results: Pts were classified according to AMI location (anterior or inferior) and the presence or absence of DET-induced ST-↑ (ST-↑ or NO ST-↑). A greater incidence of DET-induced ST ↑ was found in anterior AMI vs inferior AMI (82/175 vs 77/216, $p < 0.001$):

	Anterior AMI (175 pts)			inferior AMI (216 pts)		
	NO ST-↑ (93 pts)	p	ST-↑ (82 pts)	NO ST-↑ (139 pts)	p	ST-↑ (77 pts)
WMSI-0	1.43 ± 0.30	0.0001	1.67 ± 0.27	1.30 ± 0.18	0.0001	1.44 ± 0.27
MI (pts)	29/93	ns	30/82	48/139	ns	34/77
↑-WMSI	0.25 ± 0.14	ns	0.20 ± 0.12	0.21 ± 0.16	ns	0.18 ± 010
MV (pts)	21/93	ns	25/82	16/139	ns	11/77
↓-WMSI	0.21 ± 0.10	ns	0.17 ± 0.14	0.13 ± 0.08	ns	0.14 ± 0.09

Conclusions: In both anterior and inferior Q-wave myocardial infarction, DET-induced ST-↑ is not related to a higher prevalence and extent of MI or MV but instead to a greater extent of resting WMA.

3:15

798-6 Does the Detection of Viable Myocardium With Dobutamine Echocardiography Depend on Ventricular Function?

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Improvement in wall motion during dobutamine stress echo (DSE) has been shown to reflect myocardial viability. The specific dose to assess this effect has not been established. To determine if the optimum dose for viability assessment depends on LV function, we examined 20 patients (17 male, 3 female; age 63 ± 12) with DSE pre-CABG and compared the assessment of each segment which had improvement in regional function on resting echo at least 3 months after CABG. On DSE, the dose at which augmentation of previously abnormal segments was first noted was recorded as a positive test (+). Patients were stratified by pre-CABG ejection fraction (EF). Post-CABG wall motion was read blinded to the results of the DSE and improvement in wall motion was noted in 126 segments after CABG.

+DSE	EF < 40% (n = 9) 52/80 segments	EF ≥ 40% (n = 11) 30/46 segments	
First dose+	18 ± 13 mcg/kg/min	9 ± 9 mcg/kg/min	p < 0.07
+at ≤ 10 mcg	30/80 (38%)	25/46 (54%)	p < 0.02

Conclusions: Despite more complex or extensive wall motion abnormalities in those with decreased EF, the ability of DSE to detect viability was not impaired. However, the dose at which the augmentation occurs and, therefore, the definition of a "positive" test may vary in relation to ventricular function.